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Influence of Heating Rate on Control of Heat Loss from the Tail in Mice

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16. ABSTRACT

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Influence of heating rate on control of heat loss from the tail in mice

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GORDON, CHRISTOPHER J. *Influence of heating rate on control of heat loss from the tail in mice.* Am. J. Physiol. 244 (Regulatory Integrative Comp. Physiol. 13): R778–R784, 1983.—Although heating rate is important for stimulating thermoregulatory reflexes, it is not known if the control system differentiates between total heat gain and rate of heat gain. Exposing animals to microwaves inside a waveguide permits continuous monitoring of whole-body heat absorption. Tail skin temperature of restrained mice was recorded during whole-body exposure to 2,450-MHz microwave radiation at specific absorption rates (SAR) of either 11.5, 21.7, or 43.5 $W \cdot kg^{-1}$ and whole-body heat loads of 0.3–14 $J \cdot g^{-1}$. The integration of tail skin temperature with time, defined as the skin temperature index (STI), was measured as a function of absorbed heat load. At ambient temperatures of 20 and 25°C the STI, averaged with respect to heat load, increased significantly with SAR. Depending on SAR, the sensitivity of heat loss from the tail to microwave exposure increased 32–71% per 1°C elevation in ambient temperature. The data indicate that heat loss from the tail increases with the whole-body heat load accrued from microwave exposure. When heat loss is averaged with respect to heat load, the rate of heat absorption and ambient temperature increase the sensitivity of thermoregulatory centers that control peripheral heat loss from the tail of mice.

microwave radiation; 2,450 MHz; temperature regulation; skin temperature; specific absorption rate; heat load; vasodilation heat sensitivity; rate control

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PHYSIOLOGICAL CONTROL may be achieved with one or more of the following modes of regulation (6, 14); on-off, proportional, integrative, and rate control. Temperature homeostasis with on-off control predominates in behavioral temperature regulation (12). Proportional control has been used widely to model the physiological responses of mammals and other vertebrates to peripheral and central thermal stimulation (4). Specifically, with proportional control, the magnitude of a thermoregulatory motor output such as shivering (5), evaporative heat loss (7, 13), and peripheral (nonevaporative) heat loss (1) have been found to be proportional to the difference between temperature of the hypothalamus/preoptic area and a set point or reference temperature. Integrative control may also be important in some aspects of temperature regulation (6).

Rate control is probably very important in the control of body temperature as well as other physiological parameters (14). For example, rapid shifts in core temperature induce relatively large changes in metabolic heat production (9). Sweating in humans can be quickly inhibited with a small but rapid drop in skin temperature (15).

One limitation of past experiments is that as the rate of temperature change is altered there is a concomitant shift in total heat content of the tissues. Without accurate dosimetry it is difficult to discern if the system is responding to the rate of change in heat absorption (i.e., derivative of temperature with time) or to the overall shift in total absorbed heat (i.e., change in temperature). Therefore, to determine whether temperature regulation operates with rate control and/or proportional control it is necessary to expose the animal to various rates of heating and to measure the activity of a motor response that is normalized to the total thermal load absorbed during heat exposure.

A waveguide microwave exposure system is amenable for delivering exact whole-body heat loads at specific absorption rates (3, 8). With the recent development of temperature probes that do not perturb a microwave field, as is encountered with conventional metallic probes, it is possible to continuously monitor the temperature of mammals during microwave exposure.

Vasomotor control in the hairless tail of rodents is crucial for the dissipation of excess body heat. The rat will increase blood flow to the tail three- to fourfold when ambient temperature exceeds a threshold level of approximately 27°C (11). Thus, monitoring temperature of the tail probably gives a good representation of total peripheral heat loss when a rodent is challenged with a heat load from microwave exposure. By averaging peripheral heat loss with respect to the absorbed heat load then, if the control system employs rate sensitivity, heat loss should increase at higher heat rates. In the present study a function proportional to heat loss from the tail, normalized to the total absorbed heat load, was measured in mice exposed to 2,450-MHz microwave radiation under varying rates of absorption.

MATERIALS AND METHODS

The animals used in this study were 4-mo-old male A/J mice with a mean body weight of 26.4 ± 0.5 (SE) g. The mice were maintained at 22°C under a 12:12 L/D photoperiod.

The mice were exposed to 2,450-MHz microwave radiation inside a rectangular waveguide (5.8 × 11.2 × 30.5 cm). The microwave exposure system permitted the continuous measurement of absorbed whole-body energy.

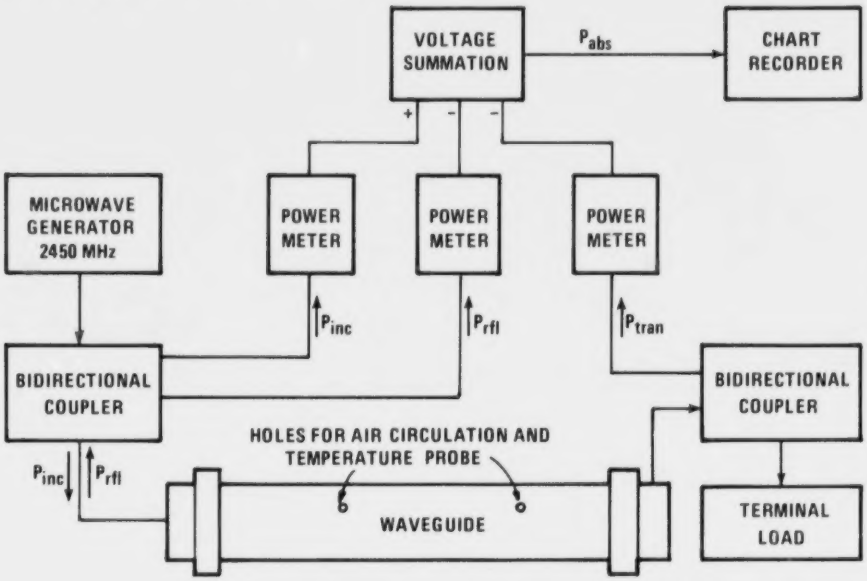

FIG. 1. Diagram of 2,450-MHz microwave exposure system. Waveguide, excluding electrical connectors, is immersed in thermostatically controlled water bath maintained at 20 or 25°C.

The schematic in Fig. 1 shows the path of microwaves through the exposure system. During microwave exposure there is a small but constant loss of power due to leakage and attenuation through the cables, electrical connectors, and animal restrainer (total loss = 16%). When biological material, which has a high electrical resistivity, is placed inside the waveguide, a large drop in power will be detected on the output of the waveguide (e.g., P_{tran}, Fig. 1). By measuring the incidence of the radiation entering (P_{inc}) and leaving the waveguide (P_{tran}) and the reflected power that emits from the mouse and the plastic restrainer back to the microwave generator, the amount of power absorbed by the animal can be calculated ($P_{abs} = P_{inc} - P_{tran} - P_{rfl}$). Approximately 70% of the incident power will be absorbed by a mouse restrained in a position parallel with respect to the long axis of the waveguide. Unabsorbed microwave energy is passed onto a 40-W terminal load (Fig. 1). Absorbed power is continuously recorded by summing the analog voltage outputs from the power meters (3).

The microwave exposure system provides an opportune means to vary the whole-body energy absorption (i.e., $J \cdot g^{-1}$) as well as the rate of energy absorption (i.e., $J \cdot g^{-1} \cdot s^{-1}$ or $W \cdot kg^{-1}$). Thus the interaction between total heat load and the rate of heat absorption on temperature regulatory processes can be easily assessed. For example, consider two exposure regimes, one with a specific absorption rate (SAR) of 100 $W \cdot kg^{-1}$ lasting 10 s and another at 10 $W \cdot kg^{-1}$ lasting 100 s. In both exposures the whole-body absorbed heat load is 1,000 $J \cdot g^{-1}$, however, the rate of heat absorption differs by one order of magnitude. If the magnitude of effector activity (e.g., metabolism, skin temperature, etc.) is averaged with respect to the absorbed heat load then, if the control of the effector depends on the rate of heat absorption, there should be a statistical correlation between SAR and motor activity.

Apparatus and protocol. The body of the waveguide, excluding the electrical connectors, was immersed in a water bath maintained at either 20 or 25°C (±0.5°C). Vents in the waveguide permitted the continuous circulation of dry air.

The mouse was restrained in a perforated polypropylene tube (3.5 x 1, 6.0 cm diam) secured to a Styrofoam plate. A polypropylene T-shaped tubing connector with an inner diameter of 3.8 mm was secured to the Styrofoam plate caudal to the mouse. The mouse's tail was inserted through the T connector and the tip of the tail was taped to the plate. A Vitek temperature probe (model 101) was inserted through the open side of the T connector, perpendicular to the long axis of the tail, and the temperature-sensing tip of the probe was positioned against the surface of the tail. Hence the T connector served to immobilize but not constrict the tail and maintain the tip of the temperature probe securely against the tail surface. Tail skin temperature was monitored continuously on a chart recorder.

A mouse was allowed to equilibrate inside the waveguide for 30–60 min. Without microwave exposure, tail temperatures settled at slightly above ambient. The mice were exposed singly to average whole-body SARs of 11.5 ± 0.3, 21.7 ± 0.4, or 43.5 ± 1.2 (SE) $W \cdot kg^{-1}$ for 30–900 s. So as not to cause excessive heat stress, exposure times were shortened at the high SARs. A mouse was usually given four microwave exposures over a 3- to 4-h period. At least 48 h elapsed before a mouse was given a subsequent microwave treatment. A total of five animals was used repeatedly.

The change in tail skin temperature with time was integrated with planimetry. In this study the integrated skin temperature response is defined as the skin temperature index (STI) with dimensions of °C·s. The STI is thus proportional to any increase in peripheral heat loss attributable to microwave exposure. The whole-body heat load accrued from microwave exposure, in dimensions of $J \cdot g^{-1}$, was derived by integrating SAR with time.

Statistical analyses of the data were made with a multiple linear regression analysis. Tests for significance were made for interactions between the dependent vari-

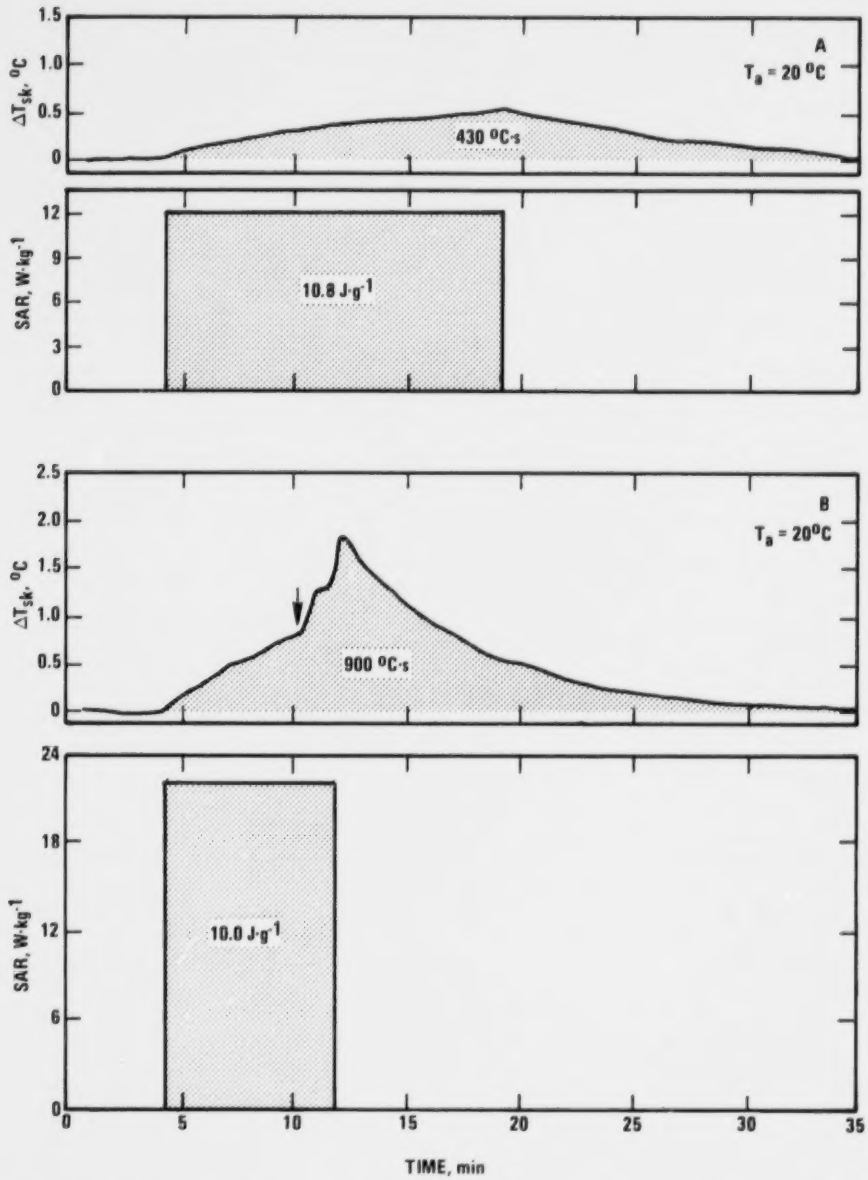

FIG. 2. Examples of changes in tail skin temperature (ΔT_{sk}) in restrained mice during exposure at 2,450-MHz microwaves at 20°C and specific absorption rate (SAR) of 12.0 $W \cdot kg^{-1}$ (*A*) and 22.3 $W \cdot kg^{-1}$ (*B*). In *A* and *B* absorbed whole-body heat load is similar but integrated skin temperature curves (skin temperature index) differ by a factor of two. *Arrow* in *B* indicates point of physiological vasodilation in tail.

able STI, the independent variables, heat load, SAR, and ambient temperature.

RESULTS

Typical changes in skin temperature of the tail following brief periods of exposure to 2,450-MHz microwaves at the low- and middle-SAR ranges are shown in Fig. 2, *A* and *B*. The area under each skin temperature curve is equal to STI. The areas under the SAR curves are equal to the whole-body heat loads accrued during microwave exposure. In Fig. 2, *A* and *B*, SARs of 12.0 $W \cdot kg^{-1}$ for 900 s and 22.3 $W \cdot kg^{-1}$ for 450 s represent nearly equal whole-body heat loads of 10.8 and 10.0 $J \cdot g^{-1}$, respectively. However, the STI at 22.3 $W \cdot kg^{-1}$ was more than double the response at 12.0 $W \cdot kg^{-1}$. In the examples (Fig. 3) recorded at an ambient temperature of 25°C, absorbing 6.4 $J \cdot g^{-1}$ at 10.8 $W \cdot kg^{-1}$ resulted in a STI of 2,949°C·s whereas absorbing 6.0 $J \cdot g^{-1}$ at 20.0 $W \cdot kg^{-1}$ led to a STI of 5,272°C·s. It is of interest to note the abrupt change in skin temperature in Figs. 2*B* and 3, *A* and *B* (see arrows) compared with the smooth continuous elevation in skin temperature in Fig. 2*A*.

The rise in skin temperature shown before the arrows in Figs. 2 and 3 is attributable to the direct heating from energy deposition on the tail. The sudden rise in temperature after the arrow is assumed to represent an active physiological vasodilation. The skin temperature where vasodilation occurred was dependent on ambient temperature (T_a) and SAR (Table 1). At 21.7 $W \cdot kg^{-1}$ the threshold was reduced by 72% by increasing T_a from 20 to 25°C. At 43.5 $W \cdot kg^{-1}$ threshold was reduced by 69% when T_a increased by 5°C. There was a significant re-

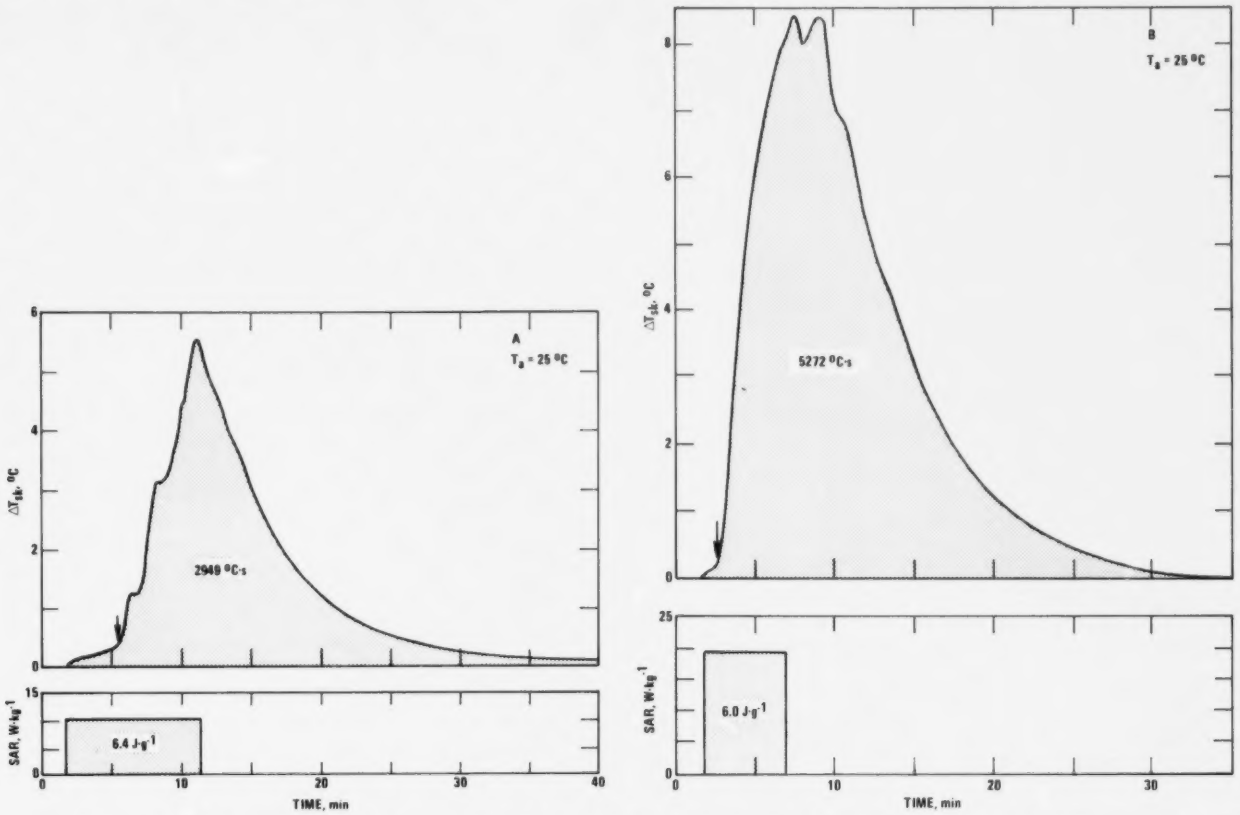

FIG. 3. Examples of change in tail skin temperature (ΔT_{sk}) in restrained mice exposed to 2,450 MHz at 25°C and specific absorption rate (SAR) of 10.6 $W \cdot kg^{-1}$ (*A*) and 20.0 $W \cdot kg^{-1}$ (*B*).

TABLE 1. *Effect of T_a and SAR on amount of increase in tail skin temperature before onset of physiological vasodilation*

T_a, °C	SAR, $W \cdot kg^{-1}$		
	11.5	21.7	43.5
20		0.84 ± 0.05 (6)*	0.59 ± 0.14 (5)*
25	0.35 ± 0.06 (7)	0.23 ± 0.04 (11)	0.18 ± 0.03 (9)†

T_a, ambient temperature; SAR, specific absorption rate. Sample size in parentheses. *$P < 0.005$ compared with same SAR at 25°C. †$P < 0.025$ compared with same temperature at 11.5 $W \cdot kg^{-1}$.

duction in threshold at 25°C when SAR increased from 11.5 to 43.5 $W \cdot kg^{-1}$.

In a dead mouse a heat load of 3.6 ± 0.1 $J \cdot g^{-1}$ delivered at SARs of 12, 22, and 40 $W \cdot kg^{-1}$ resulted in STIs of 401, 412, and 401, respectively. These data demonstrate that the rate of energy absorbed directly on the tail, when normalized to the whole-body heat load, has little effect on the overall change in STI. The STI in the dead mouse was higher than in live mice because of the very slow decay in tail skin temperature in the dead animal. In the live mouse heat from the tail is evidently being redistributed to the rest of the body.

The relationship between STI and heat load exhibited a power relationship. The effect of various heat loads on STI at SAR of 11.5 $W \cdot kg^{-1}$ and ambient temperature of 20°C is shown in Fig. 4. To make the data comparable for statistical analysis a multiple linear regression analysis was performed on double logarithimic plots of heat load vs. STI at the three average SARs and two ambient temperatures. The multiple regression analysis calculated an average dependent variable (STI) that was normalized to an averaged value of the independent variable (heat load).

With STI averaged with respect to heat load there were significant effects of SAR and ambient temperature on the control of heat loss from the tail (Fig. 5). At 20°C increasing SAR from 11.5 to 21.7 $W \cdot kg^{-1}$ promoted a 1.7-fold increase in STI. Doubling SAR to 43.5 $W \cdot kg^{-1}$ caused a 1.4-fold elevation in STI. The interaction between SAR and STI was significantly different at an ambient temperature of 25°C (Fig. 5). There was a 2.7-fold increase in STI when SAR increased from 11.5 to 21.7 $W \cdot kg^{-1}$. However, increasing SAR from 21.7 to 43.5 $W \cdot kg^{-1}$ resulted in a slight reduction (7%) in STI. In a general multiple regression analysis, which correlated the effect of the three SARs and two ambient temperatures on STI, the heat loss index was highly affected by heat load ($F = 305.4$, $P < 0.0001$), SAR ($F = 6.4$, $P < 0.0027$), and ambient temperature ($F = 26.0$, $P < 0.0001$).

DISCUSSION

The significant logarithmic correlation between heat load and STI indicates that heat loss from the tail in-

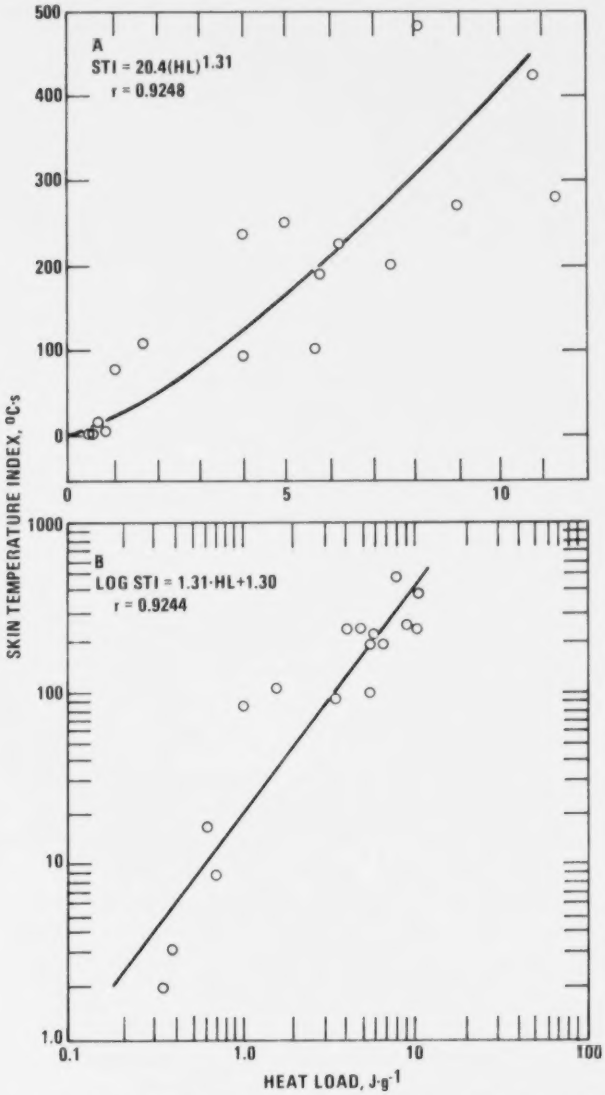

FIG. 4. Plots of skin temperature index (STI) vs. heat load at 20°C and 12 W·kg^{-1} in linear (*A*) and logarithmic dimensions (*B*). Conversion of data to linear regression in *B* permitted multiple linear statistical analysis on effects of SAR, heat load, and ambient temperature on STI.

creases as a power function with the heat load accrued from exposure to 2,450-MHz microwaves. A simple power equation adequately describes the relationship between STI and heat load from microwave exposure.

$$\text{STI} = A\ (\text{heat load})^{\alpha} \qquad (1)$$

where STI is the skin temperature index with dimensions of °C·s, α is a dimensionless exponent, and A is a constant with dimensions of °C·s·J^{-1}·g^{-1}. In the power curve fit, STI increases slowly at first and then more rapidly with increasing heat load. The power curve fit is evident because of the abrupt rise in skin temperature when heat load reached a critical level (cf., Figs. 2*B* and 3). That is, when skin temperature underwent a sharp increase at a threshold heat load STI departed from a linear relationship with head load. Hence, the power equation describes both the gradual rise in heat loss at relatively small heat loads and the abrupt increase in heat loss when heat load surpasses threshold (cf., Fig. 4).

Taking the logarithm of both sides of *Eq. 1* permits a simple linear analysis of the interaction between heat load, STI, SAR, and T_a

$$\log \text{STI} = \alpha \log \text{heat load} + \log A \qquad (2)$$

In this form α is the slope of the regression line and log A is the intercept. *Equation 2* is similar to that used to predict the response of thermoregulatory systems that operate with proportional control (4, 5, 7, 10)

$$R_1 = \alpha(T_x - T_{set}) + R_0 \qquad (3)$$

where R_0 represents base-line motor activity, α is a proportionality constant that is related to the gain of the control system, T_{set} is a set point temperature of the thermal-sensitive region that is stimulated with temperature T_x, and R_1 is the resultant effector response following temperature stimulation. Using T_x as hypothalamic temperature, *Eq. 3* has been used to predict the change in rate of metabolism and evaporative heat loss during hypothalamic thermal stimulation (5, 7, 10, 13).

In *Eqs. 2* and *3* the term α represents a coefficient of sensitivity. Past studies have shown that α in *Eq. 3* is dependent on ambient temperature (10, 13). The magnitude of T_x-T_{set} in *Eq. 3* is proportional to the error signal on the thermoregulatory system as is the magnitude of heat load in *Eq. 2*. Thus the effect of a microwave heat load on heat loss from the tail is dependent on the magnitude of α and A. For example, at the low- and medium-SAR exposures there was a relatively large increase in α when ambient temperature increased from 20 to 25°C (Table 2). However, at the high-SAR exposure the increase in α was relatively small whereas A increased substantially with increasing ambient temperature.

Because the relation between heat loss from the tail and heat load are dependent on SAR and ambient temperature, it is apparent that the control of peripheral heat loss is influenced by skin temperature and the rate of heat absorption. At an ambient temperature of 20°C, STI, averaged with respect to the microwave heat load, increased by 51% between SARs of 11.5 and 21.7 W·kg^{-1} (Fig. 5). An increase in SAR from 21.7 to 43.5 W·kg^{-1} promoted a 62% increase in STI. At 25°C STI increased by 153% between SARs of 11.5 and 21.7 W·kg^{-1}. However, STI dropped slightly when SAR increased from 21.7 to 43.5 W·kg^{-1}. Thus at the low ambient (or skin) temperature the rate sensitivity of heat loss is relatively uniform over a 32-W·kg^{-1} span in SAR. However, at 25°C the sensitivity to rate of heating is very high at the low-SAR range but it appears to saturate at high SAR levels.

The high SARs used in this study would probably not be encountered under natural circumstances. However, by using a wide SAR range, a saturation property of thermoregulatory elements involved in the control of heat loss has been demonstrated. The saturation level would appear to be quite high (at least 22 W·kg^{-1} at 25°C). Thus, rate control appears to be a very important aspect for the regulation of peripheral heat loss in mice.

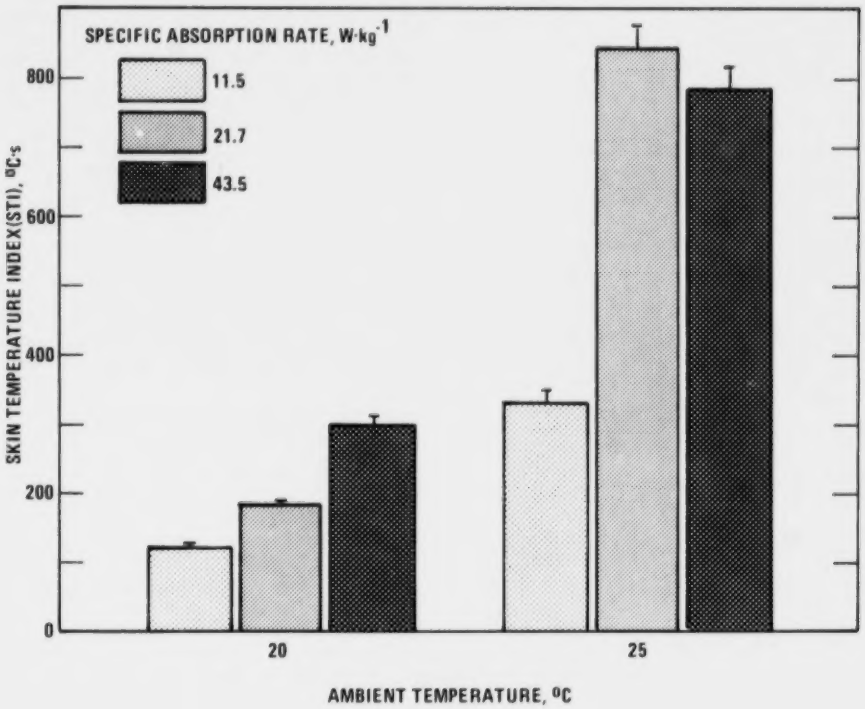

FIG. 5. Overall effect of specific absorption rate and ambient temperature on STI. Values of STI have been normalized with respect to an average heat load of 5.8 $J \cdot g^{-1}$ using multiple regression analysis. *Vertical bars* represent SEM. *n*, 80 data points from 5 animals.

TABLE 2. *Effect of T_a and SAR on the values of α and A in Eq. 1*

T_a, °C	SAR, $W \cdot kg^{-1}$	α	A	Coeff of Determination
20	11.5	1.30 ± 0.09	1.26 ± 0.14	0.8547
20	21.7	1.41 ± 0.14	1.42 ± 0.11	0.8731
20	43.5	2.04 ± 0.24	1.26 ± 0.20	0.9000
25	11.5	1.84 ± 0.25	1.42 ± 0.18	0.8233
25	21.7	1.94 ± 0.34	1.76 ± 0.24	0.7646
25	43.5	2.18 ± 0.39	1.59 ± 0.29	0.7581

Values of α and A are means ± SE. T_a, ambient temperature; SAR, specific absorption rate.

Rate control has been considered to be an important component in physiological regulation (14). Sensitivity to the rate of change of the stimulus permits a regulated system to operate with anticipatory action. Because of the time lag between heat absorption and full activation of heat-dissipating motor outputs, rate control is probably very important in temperature regulation. That is, if peripheral heat loss could change as rapidly as heat absorption then temperature homeostasis could be maintained with just proportional control. However, heat absorption from exposure to microwaves occurs instantaneously whereas several minutes are required to elevate skin temperature to its steady-state level. Evidently, the sensitivity of the controller, as estimated by the values of α and A (Table 2), must be adjusted to the SAR at or soon after the onset of microwave exposure.

Others have demonstrated the importance of rate sensitivity in temperature regulation. The control of sweating in humans is very sensitive to the rate of change in skin temperature (15). The open-loop gain of evaporative heat loss in mice is dependent on the rate of heat absorption from microwave exposure (3). The persistence of rate control in temperature regulation may be attributable to the high degree of rate sensitivity characteristic of peripheral thermal receptors (2).

Raising ambient temperature from 20 to 25°C increased the STI by 171% at 11.5 $W \cdot kg^{-1}$, 353% at 21.7 $W \cdot kg^{-1}$, and 161% at 43.5 $W \cdot kg^{-1}$ (Fig. 5). The ratio of STI to heat load is directly related to the sensitivity of the thermoregulatory elements that control peripheral heat loss. Depending on SAR the sensitivity of peripheral heat loss (i.e., STI/heat load) increased by 32 to 71% for each 1.0°C elevation in ambient temperature. Other studies have shown that the sensitivity of thermoregulatory effector activity to peripheral or central thermal stimulation is dependent on the level of ambient or skin temperature (4, 7, 10, 13).

The present study has demonstrated a principle of rate sensitivity in the control of tail skin temperature in the mouse. This study differs from many other thermal physiological studies in that the thermal stimulus was measured as rate and total heat absorption, whereas other studies have measured rate of change in temperature or steady-state temperature. Rate of change in deep-body temperature and rate of heat absorption in mice exposed to 2,450 MHz are directly related (3). Hence, measurement of total heat absorption or rate of heat absorption reflects the changes in deep-body temperature. At a constant heat load, an increase in the rate of elevation in body temperature evoked a greater vasodilatory response in the tail of the mouse. However, this phenomenon was subject to stimulus saturation as was shown at an ambient temperature of 25°C. Gordon and White (3) demonstrated a similar type of saturation of evaporative heat loss in the microwave-exposed mouse at an ambient

temperature of 32.5°C. However, saturation for evaporation occurred at a much higher SAR of approximately 160 $W \cdot kg^{-1}$ compared with just ~22 $W \cdot kg^{-1}$ for the skin temperature index. Thus, evaporative heat loss and skin temperature control in the mouse are both rate sensitive but exhibit widely different stimulus-response properties.

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This paper has been reviewed by the Health Effects Research Laboratory, US Environmental Protection Agency, and approved for publication. Mention of trade names or commercial products does not constitute endorsement or recommendation for use.

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